

# Maternal Smoking and Childhood Asthma

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**ABSTRACT.** According to a substantial literature, passive smoking by children is associated with an increased incidence of lower respiratory illness and diminished pulmonary function. The relationship between passive smoking and childhood asthma, however, is not clear. Data from the Child Health Supplement to the 1981 National Health Interview Survey were analyzed with information about 4331 children aged 0 to 5 years to study the relationship between maternal smoking and (1) the prevalence of childhood asthma, (2) the likelihood of taking asthma medication, (3) the age of onset of children's asthma, and (4) the number of hospitalizations among children with and without asthma. An odds ratio for asthma of 2.1 was shown by multivariate logistic regressions among children whose mothers smoke 0.5 packs of cigarettes or more per day compared with children of nonsmokers ( $P < .001$ ). In a similar analysis, maternal smoking of 0.5 packs per day was identified as an independent risk for children's use of asthma medication (odds ratio 1.6;  $P = .0006$ ) and for asthma developing in the first year of life (odds ratio 2.6;  $P = .0006$ ). Maternal smoking is also associated with increased numbers of hospitalizations by its association with an increased risk of asthma as well as by contributing to hospitalizations independently of a child having asthma. Among children with asthma, however, maternal smoking is not associated with increased numbers of hospitalizations. It was concluded that maternal smoking is associated with higher rates of asthma, an increased likelihood of using asthma medications, and an earlier onset of the disease. These findings have implications for renewed efforts to discourage smoking in families, especially during pregnancy and the first 5 years of children's lives. *Pediatrics* 1990;85:505-511; maternal smoking, asthma, passive smoking.

The contribution of cigarette smoke to indoor air pollution<sup>1</sup> and the adverse health consequences of

passive smoking<sup>2-5</sup> have recently come to be recognized as major public health problems. Estimates vary, but children living in temperate climates spend 60% to 80% of their time indoors<sup>6</sup> and approximately 70% of all children in the United States live in homes where there is at least one adult smoker.<sup>7,8</sup> According to a growing literature, increased childhood respiratory symptoms and altered respiratory function are associated with parental smoking. In general, it has been found in these studies that maternal smoking is more strongly correlated with children's respiratory dysfunction than is paternal smoking.<sup>9-13</sup> The most frequently offered explanations for this finding are that fathers spend less time at home than do mothers and that children spend more time with their mothers than their fathers. Hence, children are more likely to be exposed to passive smoke if their mothers smoke than if their fathers smoke. In at least two recent articles, however, it was suggested that maternal smoking during pregnancy may have independent effects on children's pulmonary structure and function.<sup>14,15</sup>

Among preschool children, the finding most frequently documented to date is an increased rate of lower respiratory infection and respiratory symptoms in children less than 2 years of age whose mothers smoke.<sup>12,13,16-18</sup> In most studies this association was shown to weaken or disappear as children grow older.<sup>12,16-18</sup> It was demonstrated in a further series of studies that maternal smoking is associated with diminished lung size<sup>19</sup> and decreased pulmonary function as measured by forced expiratory volume in 1 second, forced vital capacity, or forced expiratory flow, mid-expiratory phase among older children, thus suggesting long-term negative effects on children's pulmonary function.<sup>4,11,20-26</sup>

Although the consensus of the literature is that passive smoking is harmful to children, the rela-

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tionship between parental smoking and the prevalence and severity of childhood asthma remains unclear. There are few studies of childhood asthma and maternal smoking in which large population-based data sets were used, and none that we are aware of in which a nationwide sample was used. Previous studies have been fairly evenly divided between those in which an increased prevalence of childhood asthma or chronic wheeze associated with parental smoking<sup>8,10,22,27-29</sup> was demonstrated and those in which it was not.<sup>12,16,30-34</sup>

We analyzed data from the Child Health Supplement to the 1981 National Health Interview Survey to study the relationship between maternal smoking and (1) the prevalence of childhood asthma among children aged 0 to 5 years, (2) the likelihood of taking asthma medications prescribed by a physician, (3) the age of onset of children's asthma, and (4) the numbers of overnight hospitalizations.

## METHODS

In the National Health Interview Survey, a complex, multistage probability sampling design was used to provide a representative sample of the civilian noninstitutionalized population of the United States. In the 1981 survey there was a Child Health Supplement in which data were collected concerning one randomly chosen child in each eligible household. The supplement included 15 416 children aged birth to 17 years, of whom 4331 were aged 0 to 5 years, and contained data concerning maternal smoking. All information was derived from parent reports; there were no medical examinations of children or reviews of medical records. The interview contained a series of questions concerning family sociodemographic characteristics and a list of 59 chronic health conditions, including asthma, that children might have. Parents were asked if the index child had ever had asthma, if the asthma lasted for at least 3 months, whether the child still had asthma or if it has been cured, and how old the child was when asthma was first noticed. Children were categorized as having asthma if their parents reported that it was present at the time of the interview, had been present for more than 3 months, and had not been cured. Parents were also asked a series of questions about the age of the child at onset of asthma. In a separate series of questions, parents were asked whether the child had taken an asthma medication prescribed by a physician in the past 2 weeks. Children reported as having taken such medication for asthma were categorized as current users of asthma medications.

Questions were also asked about maternal smoking during pregnancy for all sample children aged

0 to 5 years. In other studies it has been indicated that women who smoke during pregnancy tend to continue to smoke following pregnancy.<sup>35</sup> Thus, the measure of maternal smoking used in these analyses includes both prenatal and postnatal exposure. No questions were asked about paternal smoking.

In previous studies<sup>36,37</sup> it was found that parent reports tend to overestimate the prevalence of clinically diagnosed chronic conditions; however, this overreporting tends to decline with the severity or perceived stigma of the conditions. The majority of population-based studies of childhood asthma have relied on parent reporting for the identification of children with asthma. Some authors<sup>38</sup> believe that exclusive dependence on physician reporting results in significant underreporting of childhood asthma. In one study<sup>38</sup> 96% of school-aged children with asthma could be identified by parent reporting; in another<sup>39</sup> parent reports of children's asthma were confirmed in 94% of patients,<sup>39</sup> and in another<sup>40</sup> it was shown that parent reports of childhood asthma are a good indicator of impaired ventilatory function.

## Statistical Analysis

All survey responses were weighted when we calculated means and proportions using the weights provided by the National Center for Health Statistics, which reflect the probability of selection, non-response, and poststratification adjustments. *T* tests were used to evaluate differences in means and  $\chi^2$  tests were used to measure differences in proportions. Logistic regressions were also estimated when the dependent variable was dichotomous using the PC SAS CATMOD program. The coefficient estimates can be interpreted as odds ratios associated with the predictor variable. Multivariate linear regressions were used when the dependent variable was the number of overnight hospitalizations.

Estimates of statistical significance were made assuming simple random sampling. The actual sampling design was stratified, multistage, and clustered, and the assumption of simple random sampling in this case will result in overestimates of statistical significance. We expect that design effects will be as great as 1.5. For this reason, we only discuss associations significant at the .01 level or less.

## RESULTS

As shown in Table 1, 26% of children's mothers reported smoking during pregnancy. Of these, 13% smoked less than a half-pack of cigarettes per day and 13% smoked a half-pack or more per day. Rates

and intensity of maternal smoking were substantially different for different subsets of women. Less educated women and women who report lower incomes were more likely to smoke and were more likely to smoke a half-pack of cigarettes or more per day than were more educated or more affluent women.

Asthma was reported as being present in 2.3% of children whose mothers did not smoke, 2.6% of children whose mothers smoked less than a half-pack of cigarettes per day, and 4.3% of children

whose mothers smoked a half-pack or more per day ( $P = .001$ , Table 2). In Table 3, the relative odds ratio for asthma among children aged 0 to 5 years is shown according to maternal smoking behavior. Compared with mothers who did not smoke, the odds ratio for children whose mothers smoked less than a half-pack per day is 1.1 and the comparable ratio for children whose mothers smoked a half-pack of cigarettes or more per day is 2.1 ( $P = .001$ ). When we used a multivariate analysis with a logistic regression model controlling for sex, race, presence

TABLE 1. Maternal Smoking During Pregnancy, 1981 National Health Interview Survey (n = 4536)\*

	No. of Mothers	No Smoking	Smoke < 1/2 Pack Day	Smoke ≥ 1/2 Pack Day
Race				
Black	632	74	18	5
White	3555	73	13	14
Other	144	90	9	**
Family income (\$)				
<10,000	1053	64	19	17
10,000-25,000	1868	75	13	12
25,000+	1139	80	9	10
Maternal education				
<High school	1033	62	19	19
High school	1930	71	15	14
Some college	756	84	9	7
College	596	92	5	3
All children	4331	74	13	13

\* Sample sizes will vary because of missing data. Results are given as percentages.

† Estimate not reported because number in cell is less than five observations.

TABLE 2. Prevalence of Asthma and Current Use of Asthma Medications Among Children Aged 0 to 5 Years by Maternal Smoking Status, 1981 National Health Interview Survey (n = 4331)

Maternal Smoking Status	No. of Mothers	Prevalence of Asthma (%)	P Value	% of Children Currently Using Asthma Medications	P Value
No maternal smoking	3210	2.3		0.5	
Maternal smoking < 1/2 pack/d	574	2.9	.68	*	
Maternal smoking ≥ 1/2 pack/d	547	4.8	.001	2.0	.0003
All children	4331	2.7		0.7	

\* Estimate not reported because number in cell is less than five observations.

TABLE 3. Relative Odds Ratio for Asthma and Current Use of Asthma Medications Among Children Aged 0 to 5 Years by Maternal Smoking Status, 1981 National Health Interview Survey (n = 4331)

Maternal Smoking Status	Bivariate Analysis				Multivariate Analysis*			
	Asthma	P Value	Use of Asthma Medication	P Value	Asthma	P Value	Use of Asthma Medication	P Value
No maternal smoking	1.0		1.0		1.0		1.0	
Maternal smoking < 1/2 pack/d	1.1	.68	†		1.2	.55	†	
Maternal smoking ≥ 1/2 pack/d	2.1	.001	4.1	.0003	2.1	.005	4.7	.0006

\* Control variables include sex, race, presence of both parents, family size, and number of rooms in household.

† Estimate not reported because number in cell is less than five observations.

of both biologic parents, family size, number of rooms in household, and maternal education, the odds ratios are 1.2 and 2.1, respectively ( $P = .005$ , Table 3). Family income did not add significantly to this equation at  $P < .05$ .

We examined the relationship between maternal cigarette smoking and the prevalence of children reported as using a physician-prescribed asthma medication in the past 2 weeks. Overall, 7 per 1000 children 0 to 5 years of age were reported to be using asthma medications. The prevalence of asthma medication use was strongly associated with maternal smoking; the odds of a child using asthma medication was 4.1 times greater if the mother smoked a half-pack or more of cigarettes per day compared with nonsmokers ( $P = .0003$ , Table 3). When multivariate controls were introduced to control for potential confounding variables, the odds ratio was 1.7 ( $P = .0006$ ). Control variables included sex, race, presence of both biologic parents, family size, number of rooms in the household, and maternal education. Family income did not add explanatory power to this equation.

We also estimated the association between cigarette smoking of the mother and the reported onset of asthma in the first year of the child's life. The prevalence of onset of asthma in the first year of life was 4.5% if the mother smoked a half-pack or more per day and 1.6% if she did not smoke ( $P = .0001$ ). Multivariate logistic regressions indicated an odds ratio of 2.6 if the mother smoked a half-pack or more of cigarettes per day ( $P = .0006$ , Table 4).

Because of concern that parents might mistakenly report respiratory illnesses associated with wheezing as asthma among children less than 2 years of age, we investigated the relationship between maternal smoking and asthma and use of asthma medications among children aged 2 to 5 years. With multivariate analyses, again controlling for sex, race, presence of both biologic parents, family size, number of rooms, and maternal education, we saw an odds ratio of 1.9 for asthma ( $P = .003$ ) and 3.6 for the use of asthma medications ( $P = .01$ ) for children whose mothers smoke a half-pack of cigarettes or more per day compared with children whose mothers do not smoke.

We also examined the reported number of overnight hospitalizations among children and their relationship to maternal smoking. There was a strong relationship of hospitalizations to maternal smoking (Figure). For children without asthma this relationship was highly statistically significant ( $P = .0001$ ) and changed little when controls for socioeconomic variables were introduced. For the children with asthma, the relationship between mater-

nal smoking and number of hospitalizations was not statistically significant.

## DISCUSSION

These data from the population-based Child Health Supplement to the 1981 National Health Interview Survey indicate that maternal cigarette smoking is associated with higher rates of asthma, an increased likelihood of using asthma medications, and an earlier onset of the disease among children 0 to 5 years of age, independent of a number of other potentially confounding variables. Children whose mothers smoke one half-pack of cigarettes or more per day are twice as likely to have asthma and are four times as likely to use asthma medications as are children whose mothers do not smoke. The data also demonstrate that 26% of American children live in households with mothers who report smoking during pregnancy. Currently 26% of American adults smoke (*Time*, April 18, 1988:71-90); thus, rates of prenatal and early childhood passive exposure to maternal cigarette smoke are comparable with rates of active smoking among adults in the United States.

All information in this study is based on parent reports of asthma and smoking; hence, the results should be interpreted with some caution. Questions

TABLE 4. Relative Odds Ratio for Onset of Asthma in the First Year of Life by Maternal Smoking Status, 1981 National Health Interview Survey ( $n = 4331$ )<sup>a</sup>

Maternal Smoking Status	Onset of Asthma in First Year of Life	P Value
No maternal smoking	1.0	
Maternal smoking < 1/2 pack/d	.85	.39
Maternal smoking $\geq$ 1/2 pack/d	2.6	.0006

<sup>a</sup> Control variables include sex, race, presence of both parents, family size, number of rooms in household, and maternal education.

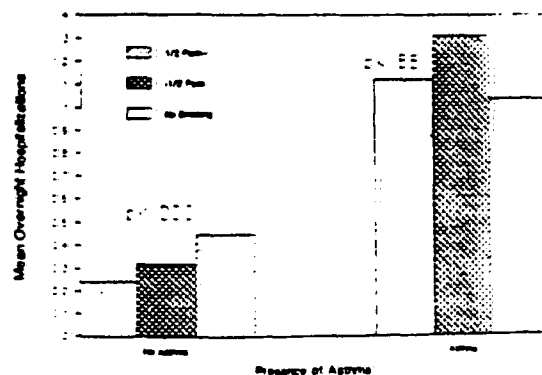


Figure. Hospitalizations by maternal smoking and asthma, children ages 0 to 5 years, 1981.

about maternal smoking were only asked in families with children aged 0 to 5 years; therefore, it is not possible to generalize these results to older children or to investigate whether more prolonged childhood exposure is associated with still higher rates of asthma or increased asthma-associated morbidity. Also, no information is available concerning maternal respiratory symptoms. In previous studies,<sup>16,41</sup> an increased incidence of respiratory symptoms was shown among adult smokers, and other studies have indicated that parent reports of their children's respiratory symptoms are influenced by their own respiratory symptoms. Physical examinations would not necessarily have resulted in more accurate reporting of children with asthma, because signs and symptoms of asthma are often intermittent and many children with asthma have normal baseline respiratory status between attacks. Similarly, information from medical records is notoriously incomplete.

The lack of a relationship between passive exposure to maternal cigarette smoke and hospitalizations among children with asthma in this study is puzzling. Although in occasional studies<sup>42</sup> there is failure to demonstrate increased bronchial reactivity among children with asthma exposed to passive smoke, in the majority of laboratory studies to date increased bronchial reactivity seems to be a fairly consistent response to passive smoking by asthmatics. The studies provide a physiologic basis for the belief that passive smoking exacerbates childhood asthma. There is surprisingly little clinical or population-based data, however, to support this belief. According to O'Connell and Long,<sup>43</sup> parents reported that their smoking aggravated their children's asthma and that the children's asthma improved when they stopped smoking. Murray and Morrison<sup>11</sup> reported 47% more symptoms among children with asthma whose mothers smoked. Tsismoyianis et al<sup>24</sup> found increased cough reported among 12- to 17-year-old nonsmoking athletes who had parents who smoked cigarettes. None of these studies, however, specify number of bed days or hospitalizations. Fergusson and Horwood<sup>12</sup> and Dodge<sup>27</sup> found no association between passive smoking and exacerbations of children's asthma. Evans et al<sup>44</sup> reported a 63% increase in emergency room use by children with asthma associated with smoking by one or more family member; however, they failed to demonstrate an association between passive smoking and days with asthma symptoms, hospitalization rates, or pulmonary function. The findings from the National Health Interview Survey also do not demonstrate an association between maternal smoking and increased hospitalizations among children with asthma. This finding must be

viewed with particular caution, however, because with only 117 children with asthma in the sample, its statistical power is low. For example, to detect a difference in hospitalization rates of 10% (with 80% power and an  $\alpha$  of .05), a sample three times larger than the present one is required.

The mechanism by which maternal smoking is associated with an increased prevalence of childhood asthma is currently not known. In most studies to date children's respiratory symptoms, asthma, and lung growth were correlated with postnatal passive smoking, but in several recent studies it was suggested that antenatal exposure to tobacco smoke might have separate, independent effects on pulmonary development and function. Collins et al<sup>14</sup> provided rat model data that suggest that maternal cigarette smoking during pregnancy is characterized by fetal lung hypoplasia with decreased lung volume and decreased numbers of alveoli. In another study<sup>15</sup> it was demonstrated that maternal smoking during pregnancy is associated with elevated cord blood IgE among newborns of nonallergic parents and a fourfold increased risk of the development of atopic disease (asthma, eczema, urticaria, or food allergy) before 18 months of age, suggesting that maternal smoking during pregnancy predisposes even low-risk infants to subsequent sensitization, probably in synergy with a subsequently acquired mucosal damage that would facilitate penetration of foreign matter. The estimate of children's exposure to cigarette smoke in the current study is crude, based on parent reporting of smoking during pregnancy. It seems reasonable to assume that for most mothers smoking habits remain relatively stable from pregnancy through early childhood and there is at least one study<sup>35</sup> to support this contention. Our data are certainly consistent with earlier findings indicating prenatal and postnatal effects on pulmonary structure and function, but it was not possible to differentiate prenatal from postnatal maternal smoking effects on the prevalence of childhood asthma.

## IMPLICATIONS

In three landmark reports by the Surgeon General<sup>2,3</sup> and the National Academy of Sciences<sup>4</sup> and the recent article by Fielding and Phenow<sup>5</sup> similar conclusions were presented about the adverse effects of passive smoking. Although passive smoking appears to present smaller risks than active smoking, the number of people injured by passive smoking is much larger than the number injured by other environmental agents that are already widely regulated. The American Academy of Pediatrics Committee on Environmental Hazards<sup>45</sup>

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has stated that passive smoking may be the most important source of environmental contamination and some believe that it is the most important environmental factor involved in the etiology of early asthma.<sup>46</sup> It is extremely unlikely that we will ever be willing or able to regulate the smoking of adults in their own homes; therefore, we must employ strategies other than coercion to help parents decrease their smoking, both for their own health as well as for their children's well-being.

The findings of this study should encourage renewed efforts to discourage smoking in families, especially during pregnancy and the first 5 years of children's lives. It is suggested that pediatricians may actually be able to help prevent childhood asthma if they can help parents stop smoking. Strategies that may be useful include explaining the environmental hazards of smoking to children, especially the association between maternal cigarette smoking and the increased risk of a child having asthma; encouraging parents not to smoke; and referring parents who smoke to smoking cessation programs. Low-cost smoking cessation programs for pregnant women have been shown to be effective,<sup>47-49</sup> but such programs have not been widely implemented or used. Two barriers to their use are the fact that insurance carriers and Medicaid generally do not pay for these programs, and physicians do not tend to refer patients to them.

The Committee on Environmental Hazards of the American Academy of Pediatrics<sup>45</sup> suggests that physicians routinely inquire about parental smoking habits when caring for children with chronic or recurrent respiratory symptoms. The data reported in this paper, when viewed in the context of other recent studies, suggest that this advice is not broad enough. Parents should be encouraged not to smoke, irrespective of their child's current respiratory status, or their smoking may result in the development of asthma in their children.

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## FANNY FARMER DIDN'T COOK UP THIS HASH

Hashing - basically an excuse to run on a surprise-filled trail and finish with beer, food and song - has reached the U.S. after years overseas, mostly in the Far East. Based on the 18th-century English school-boy game called hares and hounds, hashing was dreamed up in the 1930's by two Englishmen and an Australian living in what is now Malaysia. The trio sought to shed some pounds and shrug off a few hangovers by running around a Kuala Lumpur park.

But mere running was little dull. So the trio decided to take turns laying trails - littered with false leads - through jungles and rice fields. After navigating the course, they rewarded themselves, rather to the detriment of their original purpose, with beer in their quarters next to a club nicknamed the Hash House. (As some hashers tell it, the club barred the sweaty runners because they didn't meet its dress code.) And the hash was born.

In the ensuing decades, hashing spread among international bankers, military personnel, diplomats and others who tended to find themselves in places like Brunei with nothing to do. Now there are 80 000 hashers in more than 700 clubs in 126 countries on every continent except Antarctica.

Stout H. Following the flour is a popular sport for folks on the run. *The Wall Street Journal*. October 11, 1989.

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